

=> FILE HCAPLUS
FILE 'HCAPLUS' ENTERED AT 12:08:01 ON 27 MAY 2003
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FILE COVERS 1907 - 27 May 2003 VOL 138 ISS 22
FILE LAST UPDATED: 26 May 2003 (20030526/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> D QUE L30
L2 1457740 SEA FILE=HCAPLUS ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR MICE OR RODENT#
L3 34259 SEA FILE=HCAPLUS ABB=ON L2 AND (TEST? OR MEASUR? OR SCREEN?) AND (RODENTIC? OR TOXIC?)
L4 1 SEA FILE=REGISTRY ABB=ON 9004-34-6
L5 394825 SEA FILE=HCAPLUS ABB=ON L4 OR CORNCOB? OR COB OR COBS OR MAIZE OR ?CELLULOS?
L7 138348 SEA FILE=HCAPLUS ABB=ON L2 AND (FEED? OR DIET?)
L8 2130 SEA FILE=HCAPLUS ABB=ON L5 AND L7
L9 47045 SEA FILE=HCAPLUS ABB=ON (WEIGHT? OR WT) (2A) LOSS
L10 59149 SEA FILE=HCAPLUS ABB=ON L9 OR AD LIBITUM OR FREE? (2A) FEED?
L11 47 SEA FILE=HCAPLUS ABB=ON L8 AND L3
L12 3 SEA FILE=HCAPLUS ABB=ON L10 AND L11
L13 246 SEA FILE=HCAPLUS ABB=ON L3 AND L5
L14 3 SEA FILE=HCAPLUS ABB=ON L10 AND L13
L15 666145 SEA FILE=HCAPLUS ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM OR INTESTIN? OR GUT OR STOMACH? OR CECUM
L16 2445 SEA FILE=HCAPLUS ABB=ON L3 AND L15
L18 474846 SEA FILE=HCAPLUS ABB=ON L5 OR CORN
L19 347 SEA FILE=HCAPLUS ABB=ON L7 AND L18 AND L10
L20 71 SEA FILE=HCAPLUS ABB=ON L15 AND L19
L21 0 SEA FILE=HCAPLUS ABB=ON L20 AND (WATER? (2A) (RETEN? OR RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
L22 65 SEA FILE=HCAPLUS ABB=ON L16 AND L18
L23 2 SEA FILE=HCAPLUS ABB=ON L20 AND L3
L24 4 SEA FILE=HCAPLUS ABB=ON L22 AND (WATER? (2A) (RETEN? OR RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
L25 9 SEA FILE=HCAPLUS ABB=ON L12 OR L14 OR L21 OR L23 OR L24
L26 12 SEA FILE=HCAPLUS ABB=ON L3 AND L7 AND L10 AND L18
L27 65 SEA FILE=HCAPLUS ABB=ON L3 AND L18 AND L15
L28 7 SEA FILE=HCAPLUS ABB=ON L27 AND L10

LEVY 10/046657

page>

L29 4 SEA FILE=HCAPLUS ABB=ON L27 AND (WATER?(2A) (RETEN? OR
RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR
NECROPS?)
L30 20 SEA FILE=HCAPLUS ABB=ON L25 OR L26 OR L28 OR L29

=> FILE WPIX

FILE 'WPIX' ENTERED AT 12:08:09 ON 27 MAY 2003
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FILE LAST UPDATED: 26 MAY 2003 <20030526/UP>
MOST RECENT DERWENT UPDATE: 200333 <200333/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> NEW WEEKLY SDI FREQUENCY AVAILABLE --> see NEWS <<<

>>> SLART (Simultaneous Left and Right Truncation) is now
available in the /ABEX field. An additional search field
/BIX is also provided which comprises both /BI and /ABEX <<<

>>> PATENT IMAGES AVAILABLE FOR PRINT AND DISPLAY <<<

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES,
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>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf <<<

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=> D QUE L38

L2 1457740 SEA FILE=HCAPLUS ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR
MICE OR RODENT#
L4 1 SEA FILE=REGISTRY ABB=ON 9004-34-6
L5 394825 SEA FILE=HCAPLUS ABB=ON L4 OR CORNCOB? OR COB OR COBS OR
MAIZE OR ?CELLULOS?
L31 1658 SEA FILE=WPIX ABB=ON L2 AND (TEST? OR MEASUR? OR SCREEN?) AND
(RODENTIC? OR TOXIC?)
L32 131261 SEA FILE=WPIX ABB=ON L5 OR CORN
L33 63 SEA FILE=WPIX ABB=ON L31 AND L32
L34 339776 SEA FILE=WPIX ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM
OR INTESTIN? OR GUT OR STOMACH? OR CECUM
L35 11 SEA FILE=WPIX ABB=ON L33 AND L34
L36 2 SEA FILE=WPIX ABB=ON L33 AND (WATER?(2A) (RETEN? OR RETAIN?)
OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
L37 12 SEA FILE=WPIX ABB=ON L35 OR L36
L38 2 SEA FILE=WPIX ABB=ON L37 AND A01N?/IC

=> FILE CABAB

FILE 'CABAB' ENTERED AT 12:08:20 ON 27 MAY 2003
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FILE COVERS 1973 TO 2 May 2003 (20030502/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> D QUE L52

L2 1457740 SEA FILE=HCAPLUS ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR MICE OR RODENT#
 L4 1 SEA FILE=REGISTRY ABB=ON 9004-34-6
 L5 394825 SEA FILE=HCAPLUS ABB=ON L4 OR CORNCOB? OR COB OR COBS OR MAIZE OR ?CELLULOS?
 L31 1658 SEA FILE=WPIX ABB=ON L2 AND (TEST? OR MEASUR? OR SCREEN?) AND (RODENTIC? OR TOXIC?)
 L32 131261 SEA FILE=WPIX ABB=ON L5 OR CORN
 L33 63 SEA FILE=WPIX ABB=ON L31 AND L32
 L34 339776 SEA FILE=WPIX ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM OR INTESTIN? OR GUT OR STOMACH? OR CECUM
 L35 11 SEA FILE=WPIX ABB=ON L33 AND L34
 L36 2 SEA FILE=WPIX ABB=ON L33 AND (WATER? (2A) (RETEN? OR RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
 L39 30 SEA FILE=CABA ABB=ON L35 OR L36
 L40 0 SEA FILE=CABA ABB=ON L39 AND RODENTICID?
 L41 232245 SEA FILE=CABA ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR MICE OR RODENT#
 L43 2712 SEA FILE=CABA ABB=ON RODENTICIDES+NT/CT
 L44 380 SEA FILE=CABA ABB=ON RODENT CONTROL+NT/CT
 L45 1181 SEA FILE=CABA ABB=ON L41 AND (L43 OR L44)
 L46 219115 SEA FILE=CABA ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM OR INTESTIN? OR GUT OR STOMACH? OR CECUM
 L47 65 SEA FILE=CABA ABB=ON L45 AND L46
 L48 0 SEA FILE=CABA ABB=ON L47 AND CELLULOS?
 L49 0 SEA FILE=CABA ABB=ON L48 AND (WATER? (2A) (RETEN? OR RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
 L50 0 SEA FILE=CABA ABB=ON L39 AND (L43 OR L44)
 L51 0 SEA FILE=CABA ABB=ON L47 AND NONTOXIC?
 L52 0 SEA FILE=CABA ABB=ON L40 OR L48 OR L49 OR L50 OR L51

=> FILE AGRICOLA

FILE 'AGRICOLA' ENTERED AT 12:08:33 ON 27 MAY 2003

FILE COVERS 1970 TO 15 May 2003 (20030515/ED)

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=> D QUE L62

L2 1457740 SEA FILE=HCAPLUS ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR MICE OR RODENT#
 L4 1 SEA FILE=REGISTRY ABB=ON 9004-34-6
 L5 394825 SEA FILE=HCAPLUS ABB=ON L4 OR CORNCOB? OR COB OR COBS OR MAIZE OR ?CELLULOS?
 L31 1658 SEA FILE=WPIX ABB=ON L2 AND (TEST? OR MEASUR? OR SCREEN?) AND (RODENTIC? OR TOXIC?)

LEVY 10/046657

page>

L32 131261 SEA FILE=WPIX ABB=ON L5 OR CORN
L33 63 SEA FILE=WPIX ABB=ON L31 AND L32
L34 339776 SEA FILE=WPIX ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM
OR INTESTIN? OR GUT OR STOMACH? OR CECUM
L35 11 SEA FILE=WPIX ABB=ON L33 AND L34
L36 2 SEA FILE=WPIX ABB=ON L33 AND (WATER?(2A) (RETEN? OR RETAIN?)
OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
5 SEA FILE=AGRICOLA ABB=ON L35 OR L36
L53 707 SEA FILE=AGRICOLA ABB=ON RODENTICIDES+NT/CT
L54 0 SEA FILE=AGRICOLA ABB=ON L54 AND NONTOXIC?
L55 1 SEA FILE=AGRICOLA ABB=ON L54 AND NON(W)TOXIC?
L56 66478 SEA FILE=AGRICOLA ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR
CAECUM OR INTESTIN? OR GUT OR STOMACH? OR CECUM
L58 21 SEA FILE=AGRICOLA ABB=ON L54 AND L57
L59 0 SEA FILE=AGRICOLA ABB=ON L58 AND (WATER?(2A) (RETEN? OR
RETAIN?) OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR
NECROPS?)
0 SEA FILE=AGRICOLA ABB=ON L58 AND CELLULOS?
L60 0 SEA FILE=AGRICOLA ABB=ON L58 AND WATER?
L61 6 SEA FILE=AGRICOLA ABB=ON L53 OR L55 OR L56 OR L59 OR L60 OR
L62 L61

=> FILE BIOSIS
FILE 'BIOSIS' ENTERED AT 12:08:43 ON 27 MAY 2003
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 21 May 2003 (20030521/ED)

=> D QUE L70
L2 1457740 SEA FILE=HCAPLUS ABB=ON RAT OR RATS OR RATTUS OR MOUSE OR
MICE OR RODENT#
L4 1 SEA FILE=REGISTRY ABB=ON 9004-34-6
L5 394825 SEA FILE=HCAPLUS ABB=ON L4 OR CORNCOB? OR COB OR COBS OR
MAIZE OR ?CELLULOS?
L31 1658 SEA FILE=WPIX ABB=ON L2 AND (TEST? OR MEASUR? OR SCREEN?) AND
(RODENTIC? OR TOXIC?)
L32 131261 SEA FILE=WPIX ABB=ON L5 OR CORN
L33 63 SEA FILE=WPIX ABB=ON L31 AND L32
L34 339776 SEA FILE=WPIX ABB=ON BLOAT? OR IMPACT? OR COMPACT? OR CAECUM
OR INTESTIN? OR GUT OR STOMACH? OR CECUM
L35 11 SEA FILE=WPIX ABB=ON L33 AND L34
L36 2 SEA FILE=WPIX ABB=ON L33 AND (WATER?(2A) (RETEN? OR RETAIN?)
OR ION(W) TRANSPORT? OR POST MORTEM OR AUTOPS? OR NECROPS?)
L63 87 SEA FILE=BIOSIS ABB=ON L35 OR L36
L64 0 SEA FILE=BIOSIS ABB=ON L63 AND RODENTICID?
L65 1 SEA FILE=BIOSIS ABB=ON ?CELLULOS? AND RODENTICID?
L66 0 SEA FILE=BIOSIS ABB=ON RODENT CONTROL+NT/CT
L67 210 SEA FILE=BIOSIS ABB=ON RODENT(L) CONTROL/IT
L68 2 SEA FILE=BIOSIS ABB=ON L67 AND ?CELLULOS?
L69 0 SEA FILE=BIOSIS ABB=ON L63 AND L67
L70 2 SEA FILE=BIOSIS ABB=ON L64 OR L65 OR L66 OR L68 OR L69

=> DUP REM L30 L38 L62 L70

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FILE 'BIOSIS' ENTERED AT 12:09:04 ON 27 MAY 2003

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PROCESSING COMPLETED FOR L30

PROCESSING COMPLETED FOR L38

PROCESSING COMPLETED FOR L62

PROCESSING COMPLETED FOR L70

L71 28 DUP REM L30 L38 L62 L70 (2 DUPLICATES REMOVED)

=> D L71 1-28 ALL

L71 ANSWER 1 OF 28 HCAPLUS COPYRIGHT 2003 ACS DUPLICATE 1

AN 2002:555279 HCAPLUS

DN 137:89805

TI Rodenticide and screening method for
rodenticides

IN Dawson, William

PA Natrocell Technologies Ltd., UK

SO PCT Int. Appl., 30 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A01N065-00

ICS A01N043-16; A01N025-00; G01N037-00; G01N033-50

CC 5-5 (Agrochemical Bioregulators)

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 2002056693	A1	20020725	WO 2002-GB171	20020116	
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	GB 2374286	A1	20021016	GB 2002-943	20020116	
	US 2002160031	A1	20021031	US 2002-46657	20020116	

PRAI GB 2001-1136 A 20010117

AB A rodenticide which is nontoxic to humans, domestic animals and livestock comprises a water-retentive material as the active ingredient and a rodent attractant. Preferably the water-retentive material is cellulosic material. In certain embodiments the water-retentive material comprises .alpha.-cellulose. In one preferred

Applicant

embodiment the **cellulosic** material comprises purified **cellulose** derived from the core of the **cob** of the DK 446 **maize** hybrid or from the core of the **cob** of an agonist of the DK 446 hybrid. The unique selective **toxicity** of such **rodenticides** arises from their interference with water transport through the **gut** wall, particularly in the **cecum** (where **cellulose** is digested in **rats**). The **cecum** is vestigial in humans, who are not therefore affected by such materials. A lab. method of **screening** candidate **water-retentive** materials for **rodenticidal** activity in the field is disclosed, involving examn. of the **gut** (for **compaction** and **bloating**) and the **cecum** (for **impaction**).

ST rodenticide corncob cellulose; cecum
rodent rodenticide screening

IT Corn
(DK 446 hybrid; rodenticidal water-retentive cellulose from cob of)

IT Intestine
(cecum, rodent; use for screening of rodenticides)

IT Rat (Rattus norvegicus)
(control by water-retentive cellulose from cob of DK 446 maize hybrid)

IT Corncob
(rodenticidal water-retentive cellulose from cob of DK 446 maize hybrid)

IT Rodenticides
(water-retentive cellulose from cob of DK 446 maize hybrid)

IT 9004-34-6P, Cellulose, biological studies
RL: AGR (Agricultural use); BUU (Biological use, unclassified); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation); USES (Uses)
(rodenticidal water-retentive cellulose from cob of DK 446 maize hybrid)

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Chuhran, J; WO 9702741 A 1997
- (2) Chuhran, J; WO 9702743 A 1997
- (3) Howard, H; GB 2311464 A 1997 HCAPLUS
- (4) Howard, H; WO 9735470 A 1997

L71 ANSWER 2 OF 28 WPIX (C) 2003 THOMSON DERWENT

AN 2003-140422 [13] WPIX

DNN N2003-111565 DNC C2003-035637

TI Inhibiting tumor growth in mammalian host expressing native stimulatory lectin-like natural killer receptor NKG2D, by administering to host a composition comprising multivalent NKG2D-binding agent.

DC B04 D16 S03

IN DIEFENBACH, A; RAULET, D H
PA (DIEF-I) DIEFENBACH A; (RAUL-I) RAULET D H; (REGC) UNIV CALIFORNIA

CYC 100

PI WO 2002096459 A1 20021205 (200313)* EN 32p A61K039-395
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR

KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
 RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW
 US 2002187151 A1 20021212 (200315) A61K039-395
 ADT WO 2002096459 A1 WO 2002-US16924 20020531; US 2002187151 A1 US 2001-871491
 20010531
 PRAI US 2001-871491 20010531
 IC ICM A61K039-395
 ICS A01N061-00; A61K031-00; A61K038-00; C07K001-00; C07K002-00;
 C07K004-00; C07K005-00; C07K007-00; C07K014-00; C07K016-00;
 C07K017-00; G01N033-48
 AB WO 200296459 A UPAB: 20030224
 NOVELTY - Inhibiting (M) tumor growth in a mammalian host expressing native stimulatory lectin-like natural killer (NK)G2D receptor and determined to harbor a tumor arising in situ and containing tumor cells or determined to be predisposed to harbor the tumor, comprising administering to the host a composition of a multivalent NKG2D-binding agent, and detecting a resultant inhibition of growth of the tumor, is new.
 ACTIVITY - Cytostatic.
 The induction of therapeutic response of ligands expressing tumor cells in carcinogen induced tumors was studied. The potential of NKG2D-ligand transduced cells in the treatment of primary cancers induced by chemical carcinogenesis was examined. At the age of 50 days virgin Sprague Dawley rats were given 20 mg dimethylbenzanthracene (DMBA) dissolved at 45 deg. C in 1 ml of corn oil through a stomach catheter. Thereafter, rats were examined to monitor the outgrowth of mammary tumors at weekly intervals for the first 3 weeks, and then twice weekly until the first tumor was detected in each rat. Rats showing signs of persistent toxicity due to DMBA administration (diarrhea, fur ruffling, poor mobility) were excluded from the experiment. Palpable tumors were biopsied and isolated tumor cells were cultured and transduced with NKG2D-ligand expression vectors. At the onset of first palpable tumors, between 40 and 60 days after DMBA administration, rats were allotted to treatment (perfusion of 2 multiply 10⁶ NKG2D-ligand transduced cells) or control (corresponding untransduced cells) groups, alternating between the two. The treatment period was followed by an observation period of 28 days. Tumors were measured weekly with a caliper and tumor volumes were calculated. A single intragastric dose of 20 mg of DMBA, administered to immature, virgin rats at 50 days of age, was sufficient to induce the onset with 40-50 days of large, fast-growing tumors, localized in the mammary epithelial area, which has been classified as mammary adenocarcinomas. In carcinogen-fed rats, the onset of the first tumor nodule was detectable by palpation as early as 40 days after DMBA administration, followed by other primary tumors appearing within 7-14 days in the mammary area of the rats, reaching the maximum number of five tumors per animal. At the onset of their first palpable tumor, rats were randomly divided into two groups, one group of rats was treated with NKG2D-ligand transduced cells, and a second group of control animals was treated with equivalent untransduced cells. NKG2D-ligand transduced cells showed a powerful inhibitory activity on mammary cancer growth. From the second week of treatment (starting at day 7), up to the end of the off-therapy follow-up period, tumor burden values in the treated group were significantly lower than those in controls. At the end of treatment, the ratio between treated tumor volumes and control tumor volumes (T:C ratios) was less than 0.1. Throughout the whole experiment, no sign of toxicity from the transduced cells was detected.
 MECHANISM OF ACTION - Inhibitor of tumor growth (claimed); Enhancer

of immune response to cancer cells.

USE - (M) is useful for inhibiting tumor growth in a mammalian host expressing native NKG2D and determined to harbor a tumor arising in situ and comprising tumor cells or determined to be predisposed to harbor the tumor (claimed). (M) is effective against a wide variety of tumor types such as solid or hematological tumors, melanoma, lymphoma, sarcoma, glioma, leukemia, breast cancer or brain cancer. (M) is useful for treating neoplasia or cancer, or for enhancing the immune response to cancer cells.

Dwg.0/0

FS CPI EPI
 FA AB; DCN
 MC CPI: B04-G05; B14-H01; B14-H01A; B14-H01B; D05-H09
 EPI: S03-E14H

L71 ANSWER 3 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
 AN 2002:159699 BIOSIS
 DN PREV200200159699
 TI Rodent repellent system.
 AU Warberg, Kari G. (1)
 CS (1) 4551 78th Ave. NW., New Town, ND, 58763-9574 USA
 PI US 6337081 January 08, 2002
 SO Official Gazette of the United States Patent and Trademark Office Patents, (Jan. 8, 2002) Vol. 1254, No. 2, pp. No Pagination.
<http://www.uspto.gov/web/menu/patdata.html>. e-file.
 ISSN: 0098-1133.
 DT Patent
 LA English
 AB A rodent repellent system for repelling rodents within enclosed areas while simultaneously providing a pleasant scent. The inventive device includes a container having an opening, a drawstring within the opening of the container, cellulose fiber such as corn cob chips, and a fragrance oil. The fragrance oil preferably has a woodsy floral blend similar to potpourri. The container is preferably of a perforated material or cloth. A storage box having a lid preferably stores one or more of the containers preferably within a sealable plastic bag. The user attaches the container to a member within the vehicle such as a handle for retaining the container in a prominent position. The fragrance oil is retained by the corn cob chips and slowly released through the container. The fragrance oil provides a strong scent that repels rodents and small animals by irritating their respiratory system while simultaneously providing a pleasant scent to humans.
 NCL 424417000
 CC Pest Control, General; Pesticides; Herbicides *54600
 BC Rodentia - Unspecified 86265
 IT Major Concepts
 Equipment, Apparatus, Devices and Instrumentation; Pest Assessment
 Control and Management
 IT Chemicals & Biochemicals
 fragrance oil
 IT Methods & Equipment
 method for controlling rodents: pest control method;
 rodent repellent system: equipment
 ORGN Super Taxa
 Rodentia: Mammalia, Vertebrata, Chordata, Animalia
 ORGN Organism Name
 rodent (Rodentia): pest
 ORGN Organism Superterms

Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates;
Rodents; Vertebrates

L71 ANSWER 4 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 2002:348955 HCAPLUS
DN 137:352134
TI Consumption of an omega-3 fatty acids product, INCELL AAFA, reduced side-effects of CPT-11 (irinotecan) in **mice**
AU Hardman, W. E.; Moyer, M. P.; Cameron, I. L.
CS Pennington Biomedical Research Center, Louisiana State University, Baron Rouge, LA, 70808, USA
SO British Journal of Cancer (2002), 86(6), 983-988
CODEN: BJCAAI; ISSN: 0007-0920
PB Nature Publishing Group
DT Journal
LA English
CC 18-5 (Animal Nutrition)
AB INCELL AAFA, an omega-3 polyunsatd. fatty acid product contg. a high concn. of long chain fatty acids, was tested for its ability to ameliorate the harmful side effects of CPT-11 chemotherapy including: leukopenia, anemia, asthenia, wt. loss and liver involvement. Four groups of **mice** were fed an AIN-76 diet modified to contain: 10% wt./wt. corn oil (CO), 0% AAFA; 9% CO, 1% AAFA; 8% CO, 2% AAFA; or 7% CO, 3% AAFA. After 2 wk on the diets, half of the **mice** received CPT-11 chemotherapy (60 mg kg⁻¹ q 4 days, i.v.) the rest of the **mice** received vehicle for 2 wk. It was found that 2% AAFA in the diet of the CPT-11 treated **mice**: decreased apoptotic figures in the duodenal crypts; markedly suppressed the inflammatory eicosanoid, prostaglandin E2 in the liver; prevented liver hypertrophy; improved white blood cell counts; significantly increased red blood cell counts; decreased nos. of CPT-11 induced immature red blood cell and micronuclei in red blood cells of the peripheral blood; increased eicosapentenoic acid and docosahexaenoic acid in liver cell membranes and maintained normal grooming behavior. Thus 2% AAFA in the diet reduced the side effects of CPT-11 treatment in **mice**.
ST omega3 fatty acid INCELL AAFA irinotecan antitumor toxicity
IT Antitumor agents
 Diet
 (effect of omega-3 fatty acids product, INCELL AAFA, on side-effects of CPT-11 (irinotecan) in **mice**)
IT Fatty acids, biological studies
 RL: ADV (Adverse effect, including toxicity); FFD (Food or feed use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (polyunsatd., n-3; effect of omega-3 fatty acids product, INCELL AAFA, on side-effects of CPT-11 (irinotecan) in **mice**)
IT 474453-60-6, AAFA
 RL: ADV (Adverse effect, including toxicity); FFD (Food or feed use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (effect of omega-3 fatty acids product, INCELL AAFA, on side-effects of CPT-11 (irinotecan) in **mice**)
IT 100286-90-6, CPT-11
 RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (effect of omega-3 fatty acids product, INCELL AAFA, on side-effects of CPT-11 (irinotecan) in **mice**)

RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD

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(2003)

AN 2002:10607 AGRICOLA

DN IND23245672

TI Effect of wheat bran fiber and bran particle size on fat and fiber digestibility and gastrointestinal tract **measurements** in the **rat**.

AU Kahlon, T.S.; Chow, F.I.; Hoefer, J.L.; Betschart, A.A.

SO Cereal chemistry, July/Aug 2001. Vol. 78, No. 4. p. 481-484

Publisher: St. Paul, Minn. : American Association of Cereal Chemists, 1924-

CODEN: CECHAF; ISSN: 0009-0352

NTE Includes references

CY Minnesota; United States

DT Article

FS U.S. Imprints not USDA, Experiment or Extension

LA English

AB The effect of wheat bran (AACC hard red) and bran particle size on fat and fiber digestibility and gastrointestinal tract measurements were investigated with diets containing 5.7-10.7% dietary fiber. Fifty-six male weanling Sprague-Dawley rats were randomly assigned to four diets containing 5% cellulose (C5); 10.5% cellulose (C10); 21.5% coarse (2 mm) wheat bran (CB); or 22.2% fine (0.5 mm) wheat bran (FB) in a six-week study. Dietary fiber digestibilities were significantly different ($P < 0.05$) among treatment diets (CB > FB > C5 > C10) but there was no effect in fat digestibility among treatments. High-fiber diets fed to rats resulted in significantly greater wet and dry fecal weights than low-fiber diets. Bran diets resulted in significantly higher fecal moisture than cellulose diets. Cecum lengths increased significantly with bran diets compared with cellulose diets. The CB diet resulted in significantly higher stomach weights than with cellulose diets. Stomachs were heavier and cecal lengths were greater with bran diets than with cellulose diets; however, a high-cellulose diet resulted in increased colon weight. Except for higher fiber digestibility of coarse bran, bran particle size had no significant effects. Healthful effects of wheat bran may be associated with gastrointestinal morphology and function. Fecal bulking and decreased intestinal transit time can prevent constipation and may dilute or reduce absorption of toxic or carcinogenic metabolites, thus improving gastrointestinal health and lowering the risk of tumor development and cancer.

CC Q504 Food Composition, Field Crop Products; Q104 Food Processing, Field Crop Products; T300 Diet and Diet-related Diseases

CT animal models; cellulose; diet; dietary fat; digesta; digestibility; digestive tract; feces; feces composition; fiber; food processing quality; length; moisture content; nutrient intake; particle size; rats; weight; wheat bran

RN 9004-34-6 (CELLULOSE)

L71 ANSWER 6 OF 28 HCPLUS COPYRIGHT 2003 ACS
 AN 2001:584478 HCPLUS
 DN 135:288042
 TI Subchronic toxicity of fish oil concentrates in male and female rats
 AU Rabbani, P. Isaac; Alam, Hamida Z.; Chirtel, Stuart J.; Duvall, Robert E.; Jackson, Randolph C.; Ruffin, George
 CS Risk Assessment, HFS-308, Center for Food Safety and Applied Nutrition, Food and Drug Administration, Washington, DC, 20204, USA
 SO Journal of Nutritional Science and Vitaminology (2001), 47(3), 201-212
 CODEN: JNSVA5; ISSN: 0301-4800
 PB Center for Academic Publications Japan
 DT Journal
 LA English
 CC 18-5 (Animal Nutrition)
 Section cross-reference(s): 4
 AB The purpose of this study, second in a series, was to evaluate the effects, particularly those that may be harmful, of high-dose, long-term consumption of fish oil concs. (FOC) using male and female rats. One hundred and twenty male and 120 female rats were gavaged daily with oils and oil mixts. in a vol. equal to 0.5% body wt. (5 mL/kg/d) for 13 wk. The administered oils were corn oil, pure menhaden oil (MO), pure MaxEPA fish oil or different mixts. of corn oil with MO. The stability and the homogeneity of the dosing solns. were tested under study conditions. The animals received

isocaloric and isonitrogenous diets throughout. Food and pure water were supplied **ad libitum**. At the end of the in-life phase of the study, the animals were anesthetized with CO₂ and humanely killed by exsanguination. Blood and other tissues were prep'd. for various clin., histopathol. and lab. tests. Some beneficial effects of FOC, such as redn. in total serum cholesterol, in **rats** were confirmed. However, the authors also obsd. a significant redn. in abs. amt. of serum HDL and a significant increase in relative liver and spleen wts. in both sexes with the high dose of FOC. High doses of FOC (5 mL/kg/d) reduced serum iron and vitamin E concns. A redn. in osmotic fragility of RBC as well as an increase in RBC deformity were also obsd. in **rats** treated with high doses of FOC. These **rats** showed a significant overall increase in WBC count. It can be concluded that in **rats**, subchronic consumption of high levels of FOC can be beneficial but may also be harmful because of induction of clin. abnormalities including increased red cell deformity, increased relative liver and spleen wts., and reduced serum HDL, iron and vitamin E concns.

ST fish oil diet subchronic **toxicity**

IT Glycerides, biological studies

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(blood; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Fats and Glyceridic oils, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(fish, n-3 fatty acid-high, MaxEPA; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Fats and Glyceridic oils, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(fish; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Lipoproteins

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(high-d.; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Fats and Glyceridic oils, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(menhaden; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Fatty acids, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(polyunsatd., n-3; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Body weight

Liver

Reproductive organ

Sex

Spleen

(subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT Mineral elements, biological studies

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT 57-88-5, Cholesterol, biological studies
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (blood; subchronic **toxicity** of fish oil concs. in male and female **rats**)

IT 7439-89-6, Iron, biological studies 7723-14-0, Phosphorus, biological studies 9000-86-6, Glutamic pyruvic transaminase 9000-97-9
 9001-60-9, Lactic dehydrogenase 9001-78-9, Alkaline phosphatase 16887-00-6, Chloride, biological studies 189764-17-8, Vitamin E
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (subchronic **toxicity** of fish oil concs. in male and female **rats**)

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L71 ANSWER 7 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:5293 HCAPLUS
DN 130:193089
TI Susceptibility of roof-rats (*Rattus rattus*) to anticoagulant rodenticides baits in comparison with *Rattus norvegicus* var. *albus*
AU Youssef, Hamada M.; El-Sebai, Mohamed A.; Desheesh, Mohamed A.
CS Department of Agricultural Animal Pests, Plant Protection Research Institute, Alexandria, Egypt
SO Alexandria Science Exchange (1998), 19(3), 497-506
CODEN: ALSEEF; ISSN: 1110-0176
PB Prof. Dr. A. M. Balba Group for Soil and Water Research
DT Journal
LA English
CC 5-5 (Agrochemical Bioregulators)
AB Susceptibility of wild roof rats (*Rattus rattus*) and albino Norway rats to anticoagulant rodenticides with different formulated baits were evaluated using lab. feeding free choice test. Biochem. measurements (Hb, haematocrit, RBC's, WBC's values as well as SGOT, SGPT activities in addn. to total serum protein values) in both strains of rats were detd. The wild roof rats were

found to be less susceptible to coumatetralyl (0.0375% crushed maize and 0.045% wheat baits) than the albino Norway rats, due to the accumulation of higher dosages of active ingredient more than white rats. Chlorophacinone (0.005% crushed maize bait) was more active against both strains of rats. Hb, haematocrit and RBC's were slightly affected with the tested anticoagulant. While WBC's and sGPT were highly elevated in the blood of *Rattus rattus*. On the other hand, sGOT activities were moderately reduced in the two strains except in case of chlorophacinone which weakly elevated sGOT in *Rattus rattus*. Total serum protein was significantly reduced by the three tested anticoagulants in both strains of rats.

ST anticoagulant rodenticide rat susceptibility;

Rattus susceptibility anticoagulant rodenticide

IT Erythrocyte

Hematocrit

Leukocyte

(anticoagulant rodenticides baits effect on *Rattus rattus* and *Rattus norvegicus* var. *albus*)

IT Hemoglobins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(anticoagulant rodenticides baits effect on *Rattus rattus* and *Rattus norvegicus* var. *albus*)

IT Rodenticides

(blood coagulation-inhibiting; susceptibility of roof-rats to anticoagulant rodenticides baits in comparison with *Rattus norvegicus* var. *albus*)

IT Proteins, general, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(blood; anticoagulant rodenticides baits effect on *Rattus rattus* and *Rattus norvegicus* var. *albus*)

IT Rat (*Rattus norvegicus* albinus)

Rat (*Rattus rattus*)

(susceptibility of roof-rats to anticoagulant rodenticides baits in comparison with *Rattus norvegicus* var. *albus*)

IT 9000-86-6, GPT 9000-97-9, GOT

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(anticoagulant rodenticides baits effect on *Rattus rattus* and *Rattus norvegicus* var. *albus*)

IT 3691-35-8, Kaid 5836-29-3, Racumin

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(susceptibility of roof-rats to anticoagulant rodenticides baits in comparison with *Rattus norvegicus* var. *albus*)

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AN 1998:31121 AGRICOLA
 DN IND20815103
 TI Bait placement and acceptance by rats in macadamia orchards.
 AU Tobin, M.E.; Sugihara, R.T.; Koehler, A.E.
 AV DNAL (SB599.C8)
 SO Crop protection, Sept 1997. Vol. 16, No. 6. p. 507-510
 Publisher: Kidlington, Oxford, UK : Elsevier Science Ltd.
 CODEN: CRPTD6; ISSN: 0261-2194
 NTE Includes references
 CY England; United Kingdom
 DT Article
 FS Non-U.S. Imprint other than FAO
 LA English
 AB Black rats (*Rattus rattus*) cause extensive damage in Hawaiian macadamia (*Macadamia integrifolia*) orchards. Many growers apply rodenticides to reduce rat populations in orchards, but improper placement of bait may reduce the effectiveness of many baiting programs. We evaluated the optimum placement of bait in macadamia orchards among the three locations specified on current rodenticide labels. We placed a **non-toxic** oat bait treated with 0.75% tetracycline hydrochloride, an antibiotic that chelates with calcium in growing bones and teeth and fluoresces under UV light, in burrows, on the ground and in trees in separate orchard sections. We consistently captured the greatest percentage of marked rats (53-91%) in sections where we placed the bait in trees and the lowest percentage of marked rats (0-36%) where we broadcast bait on the ground. Placement of bait in burrows produced intermediate results (40-70%). These results suggest that broadcasting bait on the orchard floor reduces the effectiveness of rat control programs. Placing baits in trees targets rats that not only are most likely to eat the poison bait, but also are most likely to damage developing nuts.
 CC F820 Pests of Plants, Animals; L300 Animal Ecology and Behavior
 CT activity; baiting; baits; macadamia integrifolia; movement; placement; *rattus rattus*; rodent control; **rodenticides**; vertebrate pests
 ST animal activity; optimum location
 GT hawaii
 RN 64-75-5 (TETRACYCLINE HYDROCHLORIDE)
 7440-70-2 (CALCIUM)

L71 ANSWER 9 OF 28 HCPLUS COPYRIGHT 2003 ACS

AN 1997:627605 HCPLUS

DN 127:244083

TI Acute pathological and biochemical effects of 2,4,6-trinitrophenyl-N-methylnitramine (tetryl) in male **rats**

AU Reddy, Gunda; Qualls, C. W., Jr.; Hampton, A. E. G.; Yelamanchili, A.; Kim, S.

CS U.S. Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, MD, 21010-5422, USA

SO Research Communications in Pharmacology and Toxicology (1997), 2(1&2), 1-11

CODEN: RCPTFY; ISSN: 1087-1101

PB PJD Publications

DT Journal

LA English

CC 4-3 (Toxicology)

AB Male Fischer 344 rats were gavaged with 2,4,6-trinitrophenyl-N-methylnitramine (tetryl) in corn oil at 0, 500, or 1000 mg/kg. Twenty-four hours after a single treatment, hepatic and testicular drug-metabolizing enzymes, hematol., and histopathol. changes were studied. Tetryl did not produce changes in hepatic microsomal cytochrome P 450 and cytochrome b5 content but it significantly increased ethoxy and pentoxy O-dealkylase activities. Cytosolic glutathione S-transferase activity and the content of oxidized or reduced glutathione were not effected in the liver and testis. Hematol. parameters altered included an increase in the metHb, glucose, and serum urea nitrogen content and a decrease in lymphocytes. Gross necropsy revealed the presence of food contg. yellowish test substance and dark brown to red blood deposits in the stomach of treated rats even though they had been fasted for 16 h. Histopathol. changes obsd. were glycogen accumulation in the liver and focal coagulative necrosis of the gastric mucosa at the junction of the stomach and duodenum of treated rats. No other treatment-related changes were obsd. in gross or histol. evaluation.

ST tetryl acute toxicity biochem

IT Liver

IT Lymphocyte

IT Microsome

IT Stomach

IT Testis

IT (acute pathol. and biochem. effects of tetryl)

IT Hemoglobins, methemoglobins

IT RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

IT (acute pathol. and biochem. effects of tetryl)

IT Cytoplasm

IT (cytosol; acute pathol. and biochem. effects of tetryl)

IT Enzymes, biological studies

IT RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

IT (drug-metabolizing; acute pathol. and biochem. effects of tetryl)

IT Stomach

IT (mucosa; acute pathol. and biochem. effects of tetryl)

IT 479-45-8, Tetryl

IT RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)

IT (acute pathol. and biochem. effects of tetryl)

IT 9035-39-6, Cytochrome b5 9035-51-2, Cytochrome P 450, biological studies

IT 50812-37-8, Glutathione S-transferase 59793-97-4, Ethoxyresorufin O-dealkylase 96595-04-9, Pentoxyresorufin O-dealkylase

IT RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

IT (acute pathol. and biochem. effects of tetryl)
 50-99-7, Glucose, biological studies 57-13-6, Urea, biological studies
 70-18-8, GSH, biological studies 27025-41-8, GSSG
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (acute pathol. and biochem. effects of tetryl)

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AN 94:73305 AGRICOLA
 DN IND20421203
 TI Subchronic **toxicity** studies of SALATRIM structured triacylglycerols in **rats**. 2. Triacylglycerols composed of stearate, acetate, and propionate.
 AU Hayes, J.R.; Wilson, N.H.; Pence, D.H.; Williams, K.D.
 AV DNAL (381 J8223)
 SO Journal of agricultural and food chemistry, Feb 1994. Vol. 42, No. 2. p. 539-551
 Publisher: Washington, D.C. : American Chemical Society.
 CODEN: JAFCAU; ISSN: 0021-8561
 NTE Includes references
 CY District of Columbia; United States
 DT Article
 FS U.S. Imprints not USDA, Experiment or Extension
 LA English
 AB SALATRIM 23CA lot A014, SALATRIM 32CA lot A015, and SALATRIM 23SO lot A026 are members of a family of structured triacylglycerols having caloric densities of 4.5-6.0 kcal/g. **Rats** received 0%, 2%, 5% and 10% of the first two SALATRIM fats or 10% **corn** oil in the diet for 13 weeks. Body weight and feed consumption were unaffected. Minimally increased urinary phosphorus clearance occurred in 10% SALATRIM groups. Bone mineral variations and an increased incidence of renal mineralization occurred in 10% SALATRIM and **corn** oil fed **rats**. These changes appeared to be directly related to the quantity of unsaturated fatty acids in the high-fat diets. Measurements of serum and liver fat-soluble vitamin concentrations, **necropsy**, clinical pathology, organ weights, and histopathology revealed no SALATRIM-related effects. **Corn** oil produced hepatocellular vacuolation. In a supplementary short-term study, 10% dietary SALATRIM 23SO lot A026 produced no effect on selected serum transaminases. Overall, the SALATRIM fats produced no **toxicologically** significant effects.
 CC Q500 Food Composition and Quality; T300 Diet and Diet-related Diseases
 CT acetic acid; dietary fat; lipid substitutes; low fat products; **maize** oil; propionic acid; stearic acid; **toxicity**; triacylglycerols
 RN 57-11-4 (STEARIC ACID)
 64-19-7 (ACETIC ACID)
 79-09-4 (PROPIONIC ACID)
 7723-14-0 (PHOSPHORUS)
 8001-30-7 (CORN OIL)
 64706-27-0 (SALATRIM 23CA)
 64706-27-0 (SALATRIM 23SO)
 64706-27-0 (SALATRIM 32CA)

L71 ANSWER 11 OF 28 HCAPLUS COPYRIGHT 2003 ACS
 AN 1994:402906 HCAPLUS

DN 121:2906
TI Ten and ninety-day **toxicity** studies of chloropicrin in Sprague-Dawley **Rats**
AU Condie, L. W.; Daniel, F. Bernard; Olson, Greg R.; Robinson, Merrel
CS U.S. Army Dugway Proving Ground, Dugway, UT, 84022, USA
SO Drug and Chemical Toxicology (1977) (1994), 17(2), 125-37
CODEN: DCTODJ; ISSN: 0148-0545
DT Journal
LA English
CC 4-3 (Toxicology)
AB The **toxicity** of chloropicrin (CP) was assessed following its administration to **rats** via oral gavage for either 10 or 90 consecutive days at dose levels of 10, 20, 40, and 80 mg/kg and 2.8 and 32 mg/kg, resp. Control **rats** received corn oil at a dose of 1.0 mL/kg. **Toxicol.** observations include organ and body wt. measurements, necropsy and histopathol. observations, urinalysis, clin. chem. and hematol. detns. The most remarkable **toxicol.** finding in both studies was the corrosive property of CP on forestomach tissue. Inflammation, necrosis, acantholysis, hyperkeratosis and epithelial hyperplasia of the forestomach were seen in all dose groups of the 10-day study. Similar changes were detected in only the high dose group in the 90-day study. Decreased red blood cell parameters were noted in the highest dose groups in both studies, possibly due to blood loss via the damaged **stomach** lining. CP may have been aspirated into the lungs of animals in the high dose group in the ninety day study resulting in pulmonary complications leading to the deaths of 60% of the males and 80% of the females starting at week five. The 8 mg/kg dose group in the ninety day study was considered to be the no obsd. adverse effect level.
ST **toxicity** chloropicrin
IT Erythrocyte
IT Hematocrit
IT Leukocyte
IT Reticulocyte
IT Hemoglobins
IT RL: BIOL (Biological study)
IT (chloropicrin effect on)
IT Animal growth
IT (chloropicrin effect on, **toxicity** in relation to)
IT Sex
IT (chloropicrin **toxicity** in relation to)
IT Organ
IT (wt. of, chloropicrin effect on)
IT Stomach, toxic chemical and physical damage
IT (forestomach, chloropicrin **toxicity** to)
IT 76-06-2, Chloropicrin
IT RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
IT (toxicity of)
L71 ANSWER 12 OF 28 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved. (2003)
AN 1998:19151 AGRICOLA
DN IND20621181
TI Developmental effects of an environmental antiandrogen: the fungicide vinclozolin alters sex differentiation on the male **rat**.
AU Gray, L.E. Jr; Ostby, J.S.; Kelce, W.R.

CS United State Environmental Protection Agency, Research Triangle Park, NC.
SO Toxicology and applied pharmacology, Nov 1994. Vol. 129, No. 1. p. 46-52
Publisher: Orlando, Fla. : Academic Press Inc.
CODEN: TXAPPA9; ISSN: 0041-008X

NTE Includes references
CY Florida; United States
DT Article
FS U.S. Imprints not USDA, Experiment or Extension
LA English
AB In humans and **rodents**, exposure to hormonally active chemicals during sex differentiation can produce a wide range of abnormal sexual phenotypes including masculinized and defeminized females and feminized and demasculinized males. Although numerous "environmental estrogens," including pesticides, **toxic** substances (PCBs), and plant and fungal estrogens, have been shown to alter mammalian sex differentiation, similar information on environmental androgens is lacking. Recently, the fungicide vinclozolin (V) was found to inhibit sexual differentiation in male **rats** in an antiandrogenic manner. In the present study, V was administered to pregnant **rats** (po) at 0, 100, or 200 mg/kg/day in **corn** oil during the period of sex differentiation (Gestational Day 14 to Postnatal Day 3) to examine the demasculinizing effect of this fungicide more closely. In both groups of V-treated male offspring, anogenital distance was female like at birth, and nipple development was prominent at 2 weeks of age. After puberty, most of the V-treated male offspring were unable to attain intromission even though they all mounted sexually receptive females. The V-treated male offspring that appeared to achieve intromission, failed to ejaculate normally, as no sperm were found in the uterus after overnight matings. A factor in the abnormal ejaculation was that all V-treated male offspring had cleft phallus with hypospadias. In addition, a number of unusual reproductive malformations were noted when the males were **necropsied** at 1 year. Many V-treated male offspring had suprainguinal ectopic scrota/ **testes**, a vaginal pouch, epididymal granulomas, and small to absent sex accessory glands. During the study, about 25% of the V-treated males died as a result of bladder stones, hydronephrosis, or hydronephrosis, while other males displayed these lesions at **necropsy**. While some of the above malformations in male offspring can also be produced by perinatal administration of a potent estrogen, like DES, V-treated female offspring did not display any estrogen-like alterations of reproductive development or fecundity. The only change seen in the female offspring was a reduced anogenital distance during neonatal life. Our observation of perinatal-induced agenesis of the prostate and blocked **testicular** descent, a pattern of malformations nearly identical to that reported for the antiandrogen flutamide, is consistent with other recent evidence that this fungicide is an androgen-receptor antagonist.

CC H000 Pesticides, General; X380 Human Medicine, Health and Safety
CT accessory glands; ejaculation; epididymis; intromission; male animals; malformations; penis; pregnancy; progeny; **rats**; scrotum; sex differentiation; sex differentiation disorders; teratogenesis; **testes**; **toxicity**; vinclozolin
ST phallus
RN 8001-30-7 (CORN OIL)
13311-84-7 (FLUTAMIDE)
50471-44-8 (VINCLOZOLIN)

L71 ANSWER 13 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1991:466549 HCAPLUS
DN 115:66549

TI Biological and chemical characterization of metabolites of *Fusarium moniliforme* isolates
AU Hassanin, Nagawa; Gabal, M. A.
CS Dep. Nutr. Food Chem., Ain-Shams Univ., Egypt
SO Veterinary and Human Toxicology (1990), 32(6), 536-40
CODEN: VHTODE; ISSN: 0145-6296
DT Journal
LA English
CC 4-5 (Toxicology)
Section cross-reference(s): 10, 17
AB Metabolites of *F. moniliforme* isolates from different types of corn were characterized biol. and chem. The biol. assays included rat feeding, rat dermal tests, and inoculation of embryonated eggs. The metabolites were identified as diacetoxyscirpenol, ipomeanol, ipomeanine, and diplodiatoxin. The biol. tests revealed significant wt. loss in rats fed the contaminated corn for 5 wk. Hemorrhages and edema in the brain and intestine were detected in all the rats.
ST *Fusarium* metabolite toxicity
IT Toxicity
(of *Fusarium moniliforme* metabolites)
IT Mycotoxins
RL: BIOL (Biological study)
(*Fusarium moniliforme* metabolites)
IT 496-06-0, Ipomeanine 2270-40-8 41060-01-9, Diplodiatoxin 135014-96-9, 3-Ipomeanol
RL: PROC (Process)
(as metabolite of *Fusarium moniliforme*, biol. and chem. characterization of)

L71 ANSWER 14 OF 28 HCAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
AN 1990:135702 HCAPLUS
DN 112:135702
TI Absence of trichothecenes in toxigenic isolates of *Fusarium moniliforme*
AU Mirocha, C. J.; Abbas, H. K.; Vesonder, R. F.
CS Dep. Plant Pathol., Univ. Minnesota, St. Paul, MN, 55108, USA
SO Applied and Environmental Microbiology (1990), 56(2), 520-5
CODEN: AEMIDF; ISSN: 0099-2240
DT Journal
LA English
CC 10-1 (Microbial Biochemistry)
AB Thirty-four isolates of *F. moniliforme* were obtained from cereal grains collected in various parts of the world. The isolates were grown on rice and tested as a diet for toxicity to rats. Of these isolates, 53% caused death, 12% caused congestion and hemorrhage of the stomach and intestine as well as hematuria, 21% caused diarrhea, 38% caused wt. loss, and 9% were nontoxic. The cultures were tested for T-2, HT-2, neosolaniol, acetyl-T-2, T-2-tetraol, iso-T-2, diacetoxyscirpenol, monoacetoxyscirpenol, deoxynivalenol, nivalenol, fusarenone-X, 3-acetyldeoxynivalenol, 15-acetyldeoxynivalenol, zearalenone, moniliformin, fusarochromanone, fusarin-C, and wortmannin; all were neg. In addn., *F. moniliforme* NRRL A25820 was grown on corn and banana fruit as solid substrates as well as on a defined liq. medium; none of the above toxins were found. When *F. moniliforme* NRRL A25820 was incorporated into a rat diet, no toxicity was noted. Twenty-eight addnl. isolates of *E. moniliforme*, isolated from

feed assocd. with equine leukoencephalomalacia, were grown on cracked corn for 2 wk. The cultures were neg. when tested for deoxynivalenol, 15-acetyldeoxynivalenol, diacetoxyscirpenol, monoacetoxyscirpenol, nivalenol, and fusarenone X. Seventy-five percent of the isolates were toxic to ducklings, indicating the presence of a toxin other than trichothecenes. The results support the conclusion that *F. moniliforme* does not produce trichothecenes.

ST Fusarium toxigenic trichothecene absence
IT Fusarium moniliforme
(toxigenic, absence of trichothecenes in)
IT Sesquiterpenes and Sesquiterpenoids
RL: BIOL (Biological study)
(trichothecane, absence of, in toxigenic Fusarium moniliforme)

L71 ANSWER 15 OF 28 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.
(2003)
AN 89:69538 AGRICOLA
DN IND89035967
TI Utilization of four cultivars of grain amaranth growth in rats.
AU Pond, W.G.; Lehmann, J.; Clark, R.
CS USDA, ARS, Roman L. Hruska U.S. Meat Animal Research Center, Clay Center, NE
AV DNAL (RC620.A1N8)
SO Nutrition reports international, May 1989. Vol. 39, No. 5. p. 1081-1089
Publisher: Stoneham, Mass. : Butterworth.
CODEN: NURIBL; ISSN: 0029-6635
NTE Includes references.
DT Article
FS U.S. Imprints not USDA, Experiment or Extension
LA English
AB Growing Sprague-Dawley male rats were used in two experiments to determine the nutritional adequacy of four cultivars of grain amaranth for growth. The amaranth cultivars (16.8, 16.0, 16.4 and 16.1% protein) were fed as the sole source of protein and energy or diluted with maize to provide 46.1% amaranth in the diet. All cultivars were fed in the ground, unheated form. A diet containing maize as the sole source of protein (10.3%) and energy was used as a control and a 16% protein maize-soybean meal diet was fed as a reference diet. Rats were fed ad libitum for 14 days in individual wire-bottom cages in a light- and temperature-controlled room. In each experiment (5 rats fed each of 10 diets in Exp. 1 and 10 rats fed each of 6 diets in Exp. 2), body weight gain, feed intake and gain to feed ratio were recorded for each rat. Three cultivars (Amaranthus hypochondriacus 1024, A. hypochondriacus 1046 and A. hypochondriacus K188) produced weight gain and feed utilization significantly greater ($P<0.01$) than maize; two cultivars (A. hypochondriacus 1046 and A. hypochondriacus 1046 and K 188) produced weight gain not significantly different from that obtained with the maize-soy reference diet, although daily feed intake was greater with the reference diet. One cultivar (A. cruentus 1011) allowed normal growth during week 1, followed by steady weight loss and decreased feed intake during week 2. Refeeding for 7 days of half the rats fed 1011 with maize-soy reference diet resulted in rapid weight recovery and absence of gross pathology of liver, kidney, stomach, spleen, adrenal, and testes at slaughter. The nature of the toxic factor

present in *A. cruentus* 1011 is unknown, but ingestion seems not associated with permanent organ damage after 2 weeks of feeding. We conclude that three of the four amaranth cultivars tested promote growth of rats superior to that obtained with maize and comparable to that obtained with a 16% maize-soy diet.

CC Q504 Food Composition, Field Crop Products; F200 Plant Breeding and Genetics
CT amaranthus caudatus; amaranthus leucocarpus; cultivars; nutritional value; rats
RN 68308-36-1 (SOYBEAN MEAL)

L71 ANSWER 16 OF 28. HCAPLUS COPYRIGHT 2003 ACS

AN 1989:422330 HCAPLUS

DN 111:22330

TI Hepatotoxicity and renal toxicity in rats of corn samples associated with field cases of equine leukoencephalomalacia

AU Voss, K. A.; Norred, W. P.; Plattner, R. D.; Bacon, C. W.

CS Richard B. Russell Agric. Res. Cent., Agric. Res. Serv., Athens, GA, 30613, USA

SO Food and Chemical Toxicology (1989), 27(2), 89-96

CODEN: FCTOD7; ISSN: 0278-6915

DT Journal

LA English

CC 17-5 (Food and Feed Chemistry)

AB A bioassay was developed to det. the potential toxicity of corn naturally contaminated with *Fusarium moniliforme*. Two groups of five male Sprague-Dawley rats were each fed one of two *F. moniliforme*-contaminated corn samples, designated CS-1 and CS-2, that were assocd. with sep. field cases of equine leukoencephalomalacia. A control group, also consisting of 5 male rats, was fed uncontaminated seed corn. All animals survived to the end of the study, and there were no apparent differences in appearance or behavior among groups. Wt. loss and irregular food consumption occurred in all groups and probably resulted from nutritional deficiencies inherent in the corn diets.

Hepatocellular degeneration, necrosis and hyperplasia as well as biliary hyperplasia were found in the test groups only and were attributed to *F. moniliforme*. Serum transaminase and alk. phosphatase activities in animals fed CS-1 and CS-2 for 4 wk were significantly increased compared with the controls, while serum bilirubin concn. was increased only in the CS-1 group. Tubular nephrosis was also present in the renal cortex of all animals fed CS-1 and CS-2. These effects may have been related to fumonisins B1 and B2, recently discovered metabolites of *F. moniliforme*, that were found in both CS-1 and CS-2. Short-term studies of this type may be useful in screening naturally-contaminated grains and other materials for hepatotoxic metabolites produced by *F. moniliforme*.

ST corn hepatotoxicity *Fusarium* toxin leukoencephalomalacia; kidney corn *Fusarium* toxin leukoencephalomalacia; fumonisin corn bioassay

IT Feed contamination

(by hepatotoxins, of corn, bioassay of)

IT Kidney, toxic chemical and physical damage
Liver, toxic chemical and physical damage

(corn contaminated with *Fusarium moniliforme* toxicity to, equine leukoencephalomalacia in relation to)

IT *Fusarium moniliforme*

(hepatotoxicity and renal **toxicity** of **corn**
contaminated with, equine leukoencephalomalacia in relation to)

IT Corn
(hepatotoxicity and renal **toxicity** of Fusarium-contaminated,
equine leukoencephalomalacia in relation to)

IT Horse
(leukoencephalomalacia of, **dietary** Fusarium-contaminated
corn assocd. with, hepatotoxicity and renal **toxicity**
in relation to)

IT Brain, disease or disorder
(leukoencephalomalacia, equine, **dietary** Fusarium-contaminated
corn assocd. with, hepatotoxicity and renal **toxicity**
in relation to)

IT 635-65-4, Bilirubin, biological studies 9001-78-9, Alkaline phosphatase
RL: BIOL (Biological study)
(of blood serum, increase of, by **dietary corn**
contaminated with Fusarium moniliforme)

IT 116355-83-0, Fumonisin B1 116355-84-1
RL: BIOL (Biological study)
(of **corn** contaminated with Fusarium moniliforme,
toxicity of, equine leukoencephalomalacia in relation to)

IT 9031-66-7, Transaminase
RL: PROC (Process)
(of serum, increase of, by **dietary corn**
contaminated with Fusarium moniliforme)

L71 ANSWER 17 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1989:626957 HCAPLUS
DN 111:226957
TI Production of fusarenone X, nivalenol, and zearalenone by Gibberella zaeae
isolates, and their **toxicity** in fibroblasts and **rats**
AU Abbas, H. K.; Mirocha, C. J.
CS Dep. Plant Pathol., Univ. Minnesota, St. Paul, MN, 55108, USA
SO Mycotoxin Research (1988), 4(2), 67-74
CODEN: MYREET; ISSN: 0178-7888
DT Journal
LA English
CC 4-5 (Toxicology)
Section cross-reference(s): 17
AB Three isolates of G. zaeae, the perfect stage of Fusarium graminearum, were
isolated from ground **corn** cultures obtained from Taiwan in 1985
and identified as G. zaeae 1.1, G. zaeae 1-5, and G. zaeae 1-7. The isolates
were grown on a solid rice medium and exts. prep'd. with 75% aq. methanol.
The exts. were examd. for **toxicity** in the following systems:
cytotoxicity to cultured normal human diploid skin fibroblasts and
mouse fibroblasts, and **toxicity** to **rats** of
unextd. cultures. The three exts. were highly cytotoxic as indicated by
the ability to cause death and disintegration of 3T3 Swiss **mouse**
fibroblasts and human diploid skin fibroblasts during 3 to 4 days in
culture. The unextd. cultures of the isolates were highly **toxic**
to **rat**, causing hemorrhage of tissues (bladder, **stomach**
, and **intestine**), uterine enlargement, small thymuses, small
spleens, wt. loss, and death. The exts. were
tested for prodn. of trichothecenes (nivalenol and fusarenone X)
and zearalenone on rice grains. Prodn. of the 3 mycotoxins was greater at
room temp. than in the cold room. Detection of the 3 mycotoxins from the
cultures was variable, ranging from 273 to 817 ppm for nivalenol, 268 to
662 ppm for fusarenone, and 162 to 1095 ppm for zearalenone at room temp.,

and 159 to 413 ppm for nivalenol, 113 to 125 ppm for fusarenone X, and 44 to 202 ppm for zearalenone in the cold room (10.degree.).

ST fusarenone X Gibberella fibroblast; nivalenol **toxicity** fibroblast; zearalenone **toxicity** fibroblast; mycotoxin Gibberella fibroblast

IT Gibberella zae
(mycotoxins prodn. by, **toxicity** of, to fibroblasts)

IT Skin, **toxic** chemical and physical damage
(mycotoxins **toxicity** to fibroblasts of, of human)

IT Fibroblast
(mycotoxins **toxicity** to, of humans and lab. animals)

IT **Toxicity**
(of mycotoxins, to human and lab. animal fibroblasts)

IT Mycotoxins
RL: PREP (Preparation)
(prodn. of, by Gibberella zae, human and lab. animal fibroblast **toxicity** in relation to)

IT 17924-92-4P, Zearalenone 23255-69-8P, Fusarenone X 23282-20-4P,
Nivalenol
RL: PREP (Preparation)
(prodn. of, by Gibberella zae, human and lab. animal fibroblast **toxicity** in relation to)

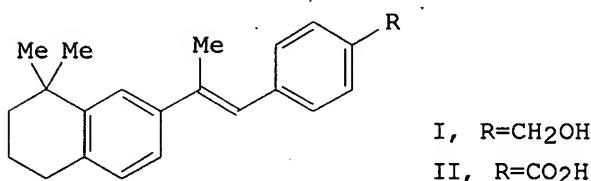
L71 ANSWER 18 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1987:472327 HCAPLUS
DN 107:72327

TI Effects of polybrominated biphenyls upon **rat** urinary protein patterns as detected by two-dimensional electrophoresis
AU Myrick, James E.; Robinson, Mary K.; Hubert, Ivey Lois; Smith, S. Jay; Hannon, W. Harry
CS Cent. Environ. Health, U.S. Dep. Health Hum. Serv., Atlanta, GA, 30333, USA
SO Archives of Environmental Contamination and Toxicology (1987), 16(5), 579-85
CODEN: AECTCV; ISSN: 0090-4341
DT Journal
LA English
CC 4-3 (Toxicology)
AB Five-month-old female **rats** were dosed by gavage with 0 or 500 mg of Fire Master BP 6 (polybrominated biphenyls, PBBs)/kg as a 5% soln. in corn oil and then maintained on normal **diet** and water **ad libitum**. Urine pools from **test** and control groups of 5 fasting **rats** were collected once a month, up to 179 days after the **test rats** were dosed. PBB-dosed animals showed significantly higher creatinine-cor. proteinuria. Two-dimensional electrophoresis (2DE) and Ag staining were used to det. differences in urinary protein patterns between **test** and control **rats**. Dosed animals excreted new proteins with higher mol. wt. and more basic isoelec. points than controls. The appearance of numerous, new proteins excreted by the PBB-dosed **rats** demonstrates the utility of 2DE for the sensitive detection of possible health effects from **toxicant** exposure.
ST brominated biphenyl urine protein; proteinuria polybrominated biphenyl
IT Urine
(proteins excretion in, after polybrominated biphenyl administration, electrophoresis for study of)
IT Proteins, biological studies
RL: BIOL (Biological study)

(metabolic disorders, proteinuria, from polybrominated biphenyls,
electrophoresis for study of)

IT 92-52-4D, Biphenyl, polybrominated biphenyls
RL: BIOL (Biological study)
(proteinuria from, electrophoresis for study of)

L71 ANSWER 19 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1987:470372 HCAPLUS
DN 107:70372
TI Preliminary toxicity profile of arotinoids SMR-2 and SMR-6 in
male B6D2F1 mice
AU Lindamood, Charles, III; Giles, Herschell D.; Hill, Donald L.
CS Kettering-Meyer Lab., South. Res. Inst., Birmingham, AL, 35255-5305, USA
SO Fundamental and Applied Toxicology (1987), 8(4), 517-30
CODEN: FAATDF; ISSN: 0272-0590
DT Journal
LA English
CC 1-6 (Pharmacology)
Section cross-reference(s): 4
GI



AB Arotinoids, which are analogs of retinoic acid (RA) and and retinol (RO) with the carbon skeleton in a rigid conformation, have more favorable therapeutic indexes relative to all-trans-RA and all-trans-RO. The purpose of this investigation was to obtain preliminary in vivo toxicity data on SMR-2 (I; analog of RO) and SMR-6 (II; analog of RA), arotinoids with promising activity for reversal of keratinization in tracheal organ culture. A preliminary toxicity study was conducted in male B6D2F1 mice with gavage of retinoids in corn oil (0.01, 0.05, and 0.1 mg/kg-day of I or II; 1,5, and 10 mg/kg-day of RA as ref. control). Due to lack of toxicity, each dose level for I and II was increased by 4-fold on day 29 of dosing. The study was terminated on day 57. Hypervitaminosis A (wt. loss, alopecia, skin scaling, and bone thinning) was induced in the mild- and high-dose SMR groups; wt.-gain depression was predominant in the high-dose RA group. The SMR compds. were .apprx.100-fold more toxic, based on wt. loss, than RA. In the SMR dose groups with hypervitaminosis A, white blood cell counts were elevated 2-4-fold; and there were microscopic lesions in skin, testes, epididymis, bone, thymus, bone marrow, peripheral lymph nodes, spleen, stomach, adrenal, and pituitary. The leukocytosis was attributed to leukopoiesis in spleen and bone marrow, which may be due to either a direct effect and/or a secondary response to a subacute inflammatory reaction in skin. Only peripheral lymph node hyperplasia was obsd. in I and RA low-dose groups. Enlarged thymus, lymph node hyperplasia, leukopoiesis in spleen and bone marrow, elevated alk. phosphatase with bone hypertrophy, and testicular degeneration were obsd. in the mid-dose RA group. The results indicate that immune stimulation may be a

primary early response to retinoids and that skin, leukopoietic tissues, reproductive organs, and bone are primary targets for retinoid toxicity.

ST arotinoid SMR2 SMR6 toxicity

IT Toxicity

(of arotinoids)

IT 89315-18-4 109791-92-6

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of)

L71 ANSWER 20 OF 28 HCAPLUS COPYRIGHT 2003 ACS

AN 1986:547953 HCAPLUS

DN 105:147953

TI Toxicology and carcinogenesis studies of 3-chloro-2-methylpropene (technical grade containing 5% dimethylvinyl chloride) (CAS No. 563-47-3) in F344/N rats and B6C3F1 mice. (Gavage studies)

CS National Toxicology Program, Research Triangle Park, NC, 27709, USA

SO Natl. Toxicol. Program Tech. Rep. Ser. (1986), 300, 196 pp.

CODEN: NTPSE3

DT Report

LA English

CC 4-6 (Toxicology)

AB Toxicol. and carcinogenesis studies of tech.-grade 3-chloro-2-methylpropene (I) [563-47-3] (contg. 5% dimethylvinyl chloride [513-37-1]), a widely used insecticide and chem. intermediate, were performed on rats and mice. In the 13-wk studies, 50%-100% mortality occurred in groups of male and female rats receiving 400 mg/kg, male rats receiving 300 mg/kg, and male and female mice receiving 500-1250 mg/kg. Inflammation and necrosis of the liver were seen in rats and mice, and necrosis of cortical tubules of the kidney was seen in mice. Based on these observations, groups of 50 male and 50 female rats were administered I in corn oil by gavage at doses of 0, 75, or 150 mg/kg, 5 days/wk for 103 wk, and groups of 50 male and 50 female mice received I at 0, 100, or 200 mg/kg on the same schedule. In the 2-yr studies, the mean body wt. of high-dose male rats was consistently 10-15% lower than that of the vehicle control group, and late in the study there was a marginal redn. in survival of high-dose male rats. Mean body wts. and survival in low-dose male rats and in both dosed groups of female rats were comparable to those of their vehicle control groups. Mean body wts. of high-dose male mice and of both dosed groups of female mice were slightly (5-9%) lower than those of the vehicle controls, whereas survival in both male and female mice was not affected by I administration. Dose-related increases in the incidence of forestomach inflammation were obsd. in male and female mice (male: vehicle control, 0/49; low dose, 9/49; high dose, 7/49; female: vehicle control, 2/50; low dose, 3/48 high dose, 9/44). Increased incidences of forestomach basal cell hyperplasia were obsd. in rats and mice of each sex. I induced forestomach squamous cell papillomas and squamous cell carcinomas in rats and mice. Invasion or metastasis of the squamous cell carcinomas to other organs was obsd. in 2 low-dose male, 3 high-dose male, and 1 high-dose female mice. Renal tubular cell adenocarcinomas (1/49), renal transitional cell carcinomas (1/49), and transitional cell papillomas (1/46) of the urinary bladder were obsd. in high-dose male rats, and renal tubular cell adenomas (1/50) and renal tubular cell

adenocarcinomas (1/50) were seen in low-dose male **rats**. These urinary tract neoplasms were not obsd. in vehicle controls. The incidences of inflammation of the nasal cavity and of nephropathy/nephrosis were greater in the 2 dosed groups than in the vehicle control groups of **rats** and **mice** of each sex. Neg. trends or lower incidences of pheochromocytomas of the adrenal gland and C-cell adenomas or carcinomas (combined) of the thyroid gland were obsd. in dosed male **rats**. Neg. trends or lower incidences of pheochromocytomas of the adrenal gland and C-cell adenomas or carcinomas (combined) of the thyroid gland were obsd. in dosed male **rats**. I was inactive or only weakly mutagenic in expts. with several strains of *Salmonella typhimurium*, but it was mutagenic in the **mouse** lymphoma L5178Y/TK+/- forward mutation assay without exogenous metabolic activation. Cytogenetics **tests** with cultured Chinese hamster ovary cells were pos. for induction of chromosomal aberrations and sister-chromatid exchanges (SCE's) in the absence of **rat** liver S9. With metabolic activation, SCE levels remained significantly elevated, but the no. of chromosomal aberrations was reduced. Under the conditions of these 2-yr gavage studies, there was clear evidence of carcinogenicity for I as shown by the increased incidences of squamous cell neoplasm in the forstomach of male and female **rats** and **mice**.

ST chloromethylpropene dimethylvinyl chloride **toxicity**; carcinogenicity chloromethylpropene dimethylvinyl chloride; mutagenicity chloromethylpropene dimethylvinyl chloride

IT Sex
(chloromethylpropene carcinogenicity and mutagenicity and **toxicity** in relation to)

IT Adrenal gland, neoplasm
Carcinoma
Kidney, neoplasm
Liver, neoplasm
Lymphoma
Mutation
Neoplasm
Papilloma
(from chloromethylpropene contg. dimethylvinyl chloride as contaminant)

IT Body weight
(loss of, from chloromethylpropene contg. dimethylvinyl chloride as contaminant)

IT Carcinoma
(adeno-, from chloromethylpropene contg. dimethylvinyl chloride as contaminant)

IT Stomach, neoplasm
(fore-, from chloromethylpropene contg. dimethylvinyl chloride as contaminant)

IT Bladder
Nose
Organ
(neoplasm, from chloromethylpropene contg. dimethylvinyl chloride as contaminant)

IT 513-37-1
RL: BIOL (Biological study)
(carcinogenicity and mutagenicity and **toxicity** of tech. grade chloromethylpropene contg.)

IT 563-47-3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(carcinogenicity and mutagenicity and **toxicity** of, contg.)

dimethylvinyl chloride as contaminant)

L71 ANSWER 21 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1986:432943 BIOSIS
DN BA82:99131
TI EFFECTS OF LEARNED FLAVOR AVOIDANCE ON GROOMING BEHAVIOR IN RATS.
AU REIDINGER R F JR; MASON J R
CS MONELL CHEMICAL SENSES CENTER, 3500 MARKET ST., PHILADELPHIA, PA 19104.
SO PHYSIOL BEHAV, (1986) 37 (6), 925-932.
CODEN: PHBHA4. ISSN: 0031-9384.
FS BA; OLD
LA English
AB In Experiment 1, rats were conditioned to avoid saccharin in tapwater by pairing it with LiCl in **carboxymethylcellulose** (CMC) applied to the fur. Conditioned flavor avoidance (CFA) of saccharin was then assessed in drinking and grooming tests. In Experiment 2, rats were given saccharin CMC on their fur and NaCl in water (or vice-versa) as conditioned stimuli in a CFA paradigm. Two-choice tests (saccharin vs. NaCl) followed in drinking and grooming contexts. In Experiment 3, rats were given saccharin CMC on one flank and vehicle (CMC only) on the other. After grooming, animals were injected with LiCl and then given 2-choice test, first between saccharin and water, then between saccharin-CMC and plain-CMC, and finally, between saccharin and water. Strong CFA was exhibited in drinking tests in all 3 experiments. This was not the case in grooming tests. Rats continued to groom when tastant was applied to only one flank (Experiment 1), and exhibited only weak CFA when a different tastant was applied to each flank (Experiments 2 and 3). We conclude that grooming can be directed to minimize the ingestion of noxious substances, but that such ingestion is not sufficiently reduced to affect the efficacy of grooming as a delivery method for unpalatable substances (e.g., **rodenticides**, chemosterilants). We speculate that grooming represents a weakness in rodents' defenses against dietary poisoning, and it might be used to deliver toxicants as part of crop protection schemes that make use of CFA.
CC Behavioral Biology - Animal Behavior *07003
Behavioral Biology - Conditioning *07005
Nervous System - Physiology and Biochemistry 20504
Psychiatry - Psychophysiology *21003
BC Muridae 86375
IT Miscellaneous Descriptors
CONDITIONING RODENT CONTROL
L71 ANSWER 22 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1979:469446 HCAPLUS
DN 91:69446
TI Effects of intubating **rats** with fractions from thermally oxidized **corn** oil and olive oil
AU Gabriel, H. G.; Alexander, J. C.; Valli, V. E.
CS Dep. Nutr., Univ. Guelph, Guelph, ON, N1G 2W1, Can.
SO Nutrition Reports International (1979), 19(4), 515-26
CODEN: NURIBL; ISSN: 0029-6635
DT Journal
LA English
CC 4-3 (Toxicology)
Section cross-reference(s): 17
AB Male weanling **rats** were maintained on Purina lab. chow **ad libitum** throughout the exptl. period, and received either 0.5 mL/day of the distillable nonurea adductable material (DNUA) of

thermally oxidized corn oil (CO) or olive oil (OO), or the same amt. of the fresh fats by stomach tube. Within 3 days, the rats receiving DNUA-OO had lost wt. and were moribund. The rats intubated with DNUA-CO gained wt. and were indistinguishable in appearance from the control animals. Addnl. urea adductions of DNUA-CO yielded DNUA-2-CO, which was comparable in toxicity to DNUA-OO. The animals in both the latter groups had severe diarrhea, seborrhea and flatulence. Pathol. examn. revealed visceral edema, gastric ulcers, and yeast infection of the gut. Histopathol. examn. of tissues showed diffuse lesions. Livers and kidneys of the 3 test groups were increased in wt. compared to their resp. controls. Extensive changes in constituent fatty acids of the neutral and polar organ lipids were obsd. Overall evaluation of the thermally oxidized samples showed that DNUA-2-CO and DNUA-OO were very toxic, whereas DNUA-CO compared favorably with the control fats.

ST corn oil thermal oxidn toxicity; olive oil thermal oxidn toxicity

IT Fats, biological studies

RL: RCT (Reactant); RACT (Reactant or reagent)
(thermal oxidn. of, toxicity in relation to)

IT Corn oil

Olive oil

RL: BIOL (Biological study)
(thermally-oxidized, toxicity of)

L71 ANSWER 23 OF 28 HCAPLUS COPYRIGHT 2003 ACS

AN 1977:512696 HCAPLUS

DN 87:112696

TI Myocardial lesions in rats fed rapeseed oil. I. Influence of strain of rats

AU Hulan, H. W.; Kramer, J. K. G.; Corner, A. H.

CS Res. Branch, Agric. Canada, Ottawa, ON, Can.

SO Canadian Journal of Physiology and Pharmacology (1977), 55(2), 258-64
CODEN: CJPPA3; ISSN: 0008-4212

DT Journal

LA English

CC 4-6 (Toxicology)

Section cross-reference(s): 17

AB The influence of strain of rat on the development of myocardial lesions was investigated in an expt. which included 2 factors: strain (Wistar, Sherman, Chester Beatty (Hooded), and Sprague-Dawley from 2 sources designated Sprague-Dawley A, Sprague-Dawley C) and diet (5% corn oil, 20% corn oil, and 20% Brassica napus var. Zephyr rapeseed oil). Groups of 30 rats, housed 2 per cage, from each of 3 different strains of rats and groups of 10 rats, housed 2 per cage, from one strain of rats (Hooded) were fed semisynthetic diets contg. the test oils for 16 weeks on an ad libitum basis.

Rats of the Hooded strain consumed considerably less feed and grew at a significantly slower rate than did rats of the Sprague-Dawley C strain, which in turn ate less and grew significantly slower than rats of the other 3 strains. No marked differences were obsd. in the fatty acid compn. of total cardiac lipids among strains. Rats from all strains except Hooded fed the diet contg.

20% Zephyr RSO had a significantly higher incidence of myocardial lesions than rats fed the 20% corn oil diet which in turn had a significantly higher incidence than rats fed 5% corn oil. Similarly, significantly more Sprague-Dawley C

rats had myocardial lesions than Sprague-Dawley A rats regardless of diet. None of the Hooded rats fed the diet contg. 20% Zephyr RSO developed myocardial lesions whereas all other strains fed this diet developed a high incidence of myocardial lesions.

ST myocardial lesion rapeseed oil; heart damage rapeseed oil
 IT Heart, toxic chemical and physical damage
 (myocardial lesions from rapeseed oil in, rat strains in relation to)
 IT Rape oil
 RL: BIOL (Biological study)
 (myocardial lesions from, rat strains in relation to)

L71 ANSWER 24 OF 28 HCAPLUS COPYRIGHT 2003 ACS

AN 1972:139084 HCAPLUS

DN 76:139084

TI Nutritive protein from cellulose

IN Srinivasan, Vadake R.; Callihan, Clayton D.

PA Louisiana State University Foundation

SO U.S., 4 pp.

CODEN: USXXAM

DT Patent

LA English

IC C12D

NCL 195033000

CC 16 (Fermentations)

Section cross-reference(s): 17, 18

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 3627095	A	19711214	US 1969-847719	19690805
PRAI US 1969-847719		19690805		

AB Nutritive protein is prep'd. from cellulose by cultivating a cellulase-elaborating microorganism and Alcaligenes faecalis on delignified cellulose under submerged aerobic conditions at pH 5-9 and temp. 25-65.degree.. Thus, 760 parts of sugarcane bagasse was slurried in 8% NaOH for 1 hr at room temp. Excess liq. was removed and the residual solid was spread in a thin layer on a wire screen and heated in a hot air oven at 110.degree. for 15 min. The partially dried material was dild. to 193.1 l. with a sterile aq. soln. contg. NaCl 900, (NH4)2SO4 194, KH2PO4 97, K2HPO4 97, MgSO4 19.4, CaCl2 19.4, and yeast ext. 60 g. Sufficient HCl to neutralize and to keep the soln. at the neutral point was added. The mixt. was boiled 3 hr and air was introduced slowly for cooling. When the temp. reached 32.degree., 1 l. of inoculum contg. Cellulomonas species ATCC No. 21399 and A. faecalis ATCC No. 21400 was added. With aeration at 34 l./min, fermentation was carried on for 90.25 hr at 32.degree.. After sepg. unreacted cellulose and harvesting the bacterial cells produced, 738 g (97.2%) of the initial insol. cellulose had been solubilized and a yield of 370 g of bacterial cells was obtained. The cells contained 50.2% protein. The protein, hydrolyzed in 6N HCl for 22 hr at 110.degree., contained arginine 5.3, histidine 2.3, isoleucine 6.0, leucine 10.0, lysine 6.5, methionine 2.0, phenylalanine 5.8, tyrosine 2.7, threonine 8.3, and valine 5.6%. The digestibility of the protein was detd. by feeding weanling rats ad libitum over a 10-day period with a protein-free diet and the same diet with 10, 20, or 40% of the produced protein. All the rats receiving 20% or more protein in the diet showed a wt. increase, while those on the

protein-free diet showed a net wt. loss. No toxicity occurred at any feeding level.

ST protein nutrition cellulose Alcaligenes

IT Cellulomonas (cultivation of Alcaligenes faecalis and, on cellulose)

IT Alcaligenes faecalis (cultivation of Cellulomonas and, on cellulose)

IT Bagasse (fermentation of, protein manuf. by)

IT Proteins
RL: BIOL (Biological study)
(single-cell, from cellulose)

IT 9004-34-6, biological studies
RL: BIOL (Biological study)
(fermentation of, protein manuf. by)

L71 ANSWER 25 OF 28 HCPLUS COPYRIGHT 2003 ACS
AN 1967:103813 HCPLUS
DN 66:103813
TI Relation between cerebral sulphydryl concentration and the central action of some thio compounds
AU Cascio, Giovanni; Palazzoadriano, Mario; Madonia, Paolino
CS Univ. Palermo, Sicily, Italy
SO Biochimica e Biologia Sperimentale (1966), 5(1), 3-9
CODEN: BBSPAJ; ISSN: 0006-2995
DT Journal
LA Italian
CC 15 (Pharmacodynamics)
AB The effect of cysteamine (I), BAL (II), cysteamine-N-acetic acid (III), and cysteine (IV), alone or in assocn. with substances capable of influencing oxidn.-redn. processes (vitamin B12 (V) and dinitrophenol (VI)), on behavior and cerebral and liver sulphydryl (SH) concn. was studied in rats and doves, maintained under standard conditions and diets, the doves being given maize and water ad libitum. The compds. were injected into the 24-hr. fasted animals, i.p. in the rats and i.m. in the doves. When V and VI were used, they were administered 30 min. before the thio compds. Thirty min. after injection of the thio compds. the animals were killed by decapitation, bled, and the brain and liver removed. Portions of 300 mg. were homogenized in water and the SH detd. iodometrically (Merville, et al., CA 55, 9531i). With I the ED100 was 200 mg./kg. and the min. convulsant level of SH was a 24% increase above normal. Below this dose brain and liver SH levels were not significantly affected, even at 180 mg./kg. At 200 mg./kg. if the rats were killed before onset of convulsions, cerebral SH was not affected, but if killed during convulsions, brain SH markedly increased (33%) while liver SH was not affected. Pretreatment with V or VI (10 and 20 mg./kg. resp.) which alone did not modify the SH levels, produced convulsions and increases in cerebral SH with the normally non-convulsant dose of 180 mg./kg. In doves I produced no convulsions even at 300 mg./kg. and did not affect cerebral free SH with or without V or VI. II gave similar results; 45 mg./kg. had no effect, 80 mg./kg. produced convulsions and a 33% rise in cerebral and liver SH in rats. With 10 mg./kg. V, 45 mg./kg. produced the same effect as 80 mg./kg. used alone. III even in toxic doses showed a depressive action in all species tested and in rats at 800 mg./kg. did not appreciably affect cerebral SH but increased liver SH distinctly. Pretreatment with V or VI did not modify the effects. IV at 500 mg./kg. produced no appreciable effect but 1000

mg./kg. produced depressive symptoms with 16% rise in cerebral SH. The symptoms lasted 2-3 hrs., then cleared, leaving the animal apparently normal. Pretreatment with V had no effect but that with VI provoked excitatory phenomena with 52% rise in cerebral SH. It is presumed that the convulsant capacity depends on the ratio of velocity of passage through the blood-brain barrier and the rate of oxidn. of nervous tissue and that V and VI accentuate the convulsant power by favoring the permanence of a state of redn. and permitting the concn. of SH in the central nervous system to reach concns. sufficiently higher than normal.

11 references.

ST SULFHYDRYLS BRAIN; BRAIN SULFHYDRYLS; LIVER SULFHYDRYLS; THIO COMPDS TISSUE SULFHYDRYLS; CONVULSANTS THIO COMPDS; VITAMIN B12 CONVULSIONS

IT Mercapto group
(in brain after thio compd. administration, behavior and)

IT Brain, composition
Liver, composition
(mercapto group in, thio compd. effect on)

IT Behavior
(thio compd. effect on, mercapto groups in brain and)

IT 51-28-5, biological studies
RL: BIOL (Biological study)
(behavior and mercapto group in brain after treatment with, thio compds. and)

IT 52-90-4, biological studies 59-52-9 60-23-1 3724-83-2
RL: BIOL (Biological study)
(behavior and mercapto groups in brain after treatment with)

IT 68-19-9, biological studies
RL: BIOL (Biological study)
(behavior in mercapto groups in brain after treatment with, thio compds. and)

L71 ANSWER 26 OF 28 HCAPLUS COPYRIGHT 2003 ACS
AN 1964:48411 HCAPLUS
DN 60:48411
OREF 60:8542e-h
TI Toxicological and hygienic characterization of new chlorinated insecticides and food products contaminated by them
AU Serebryanaya, S. G.
SO Gigiena i Toksikol. Novykh Pestitsidov i Klinika Otravlenii, Dokl. 2-oi [Vtoroi] Vses. Konf. (1962) 124-33
DT Journal
LA Unavailable
CC 69 (Toxicology, Air Pollution, and Industrial Hygiene)
AB The effect of dichlorodiphenyl dichloroethane (I),
diethyldiphenyl dichloroethane (II), dimethoxydiphenyltrichloroethane
ne (III), and dichlorodiphenyltrichloroethane (IV) on the behavior of
animals, body wt., duration of intoxication, reproduction and succeeding
generations, morphological compn. of blood and changes in internal organs,
phys.-chem. changes in urine, glycemia, carbohydrate metabolism in liver,
Thunberg's test, and changes of relative wt. of organs was
examd. in rats, mice, and cats. The minimal
toxic dose, and L.D.min. of I, II, III, and IV was 1000, 2000,
3000, and 150 mg./kg. and 3000, >4000, >5000, and 300 mg./kg., resp., for
rats; -, 1000, -, and 150 mg./kg. and 3000, >4000, 4000, and 200
mg./kg., resp., for cats; and 2000, 2000, 2000, and 150 mg./kg. and 4000,
>4000, -, and 200 mg./kg. for mice. I, II, and III had no
cumulative effect. During 11 months of daily administration of I, II,
III, and IV, the minimal physiol. active doses were 10, 25, 10, and 0.2

mg./kg. for rats and 5, 10, 10, and 0.1 mg./kg. for cats. I, II, and III, when administered daily to pregnant animals or during lactation, had no effect on the 2nd generation. IV was toxic to lactating animals. After chronic I, II, III, and IV intoxication, anorexia, loss of wt., hypohemoglobinemia, leukocytosis, and tremor were observed. The glycemic curves had a diabetic character only after III or IV application. IV caused a decrease of dehydrogenation activity of the liver. I and III gave a similar, but smaller effect, while II potentiated this function. IV caused an increase of the relative wt. of liver, while I, II, and III did not. The max. permissible amts. in food products were established as 7 mg./kg. in fruit and 3.5 mg./kg. in corn for I, 14 and 7 mg./kg. for II, and 14 mg./kg. for all products for III.

IT Blood
Nervous system
(chlorinated insecticide effect on)
IT Insecticides
(chlorinated, toxicology of)
IT Liver
(effect of chlorinated insecticides on)
IT Food
(insecticide residues in)
IT Field theories
(symmetry, boson decay probabilities and)
IT Ethane, 1,1,1-trichloro-2,2-bis(p-methoxybenzyl)-
(toxicology of)
IT 50-99-7, Blood sugar
(chlorinated-insecticide effect on)
IT 72-54-8, Ethane, 1,1-dichloro-2,2-bis(p-chlorophenyl)-
(toxicity to Amblyseius hibisci and other phytoseiid mites,
toxicology of)
IT 50-29-3, Ethane, 1,1,1-trichloro-2,2-bis(p-chlorophenyl)- 72-56-0,
Ethane, 1,1-dichloro-2,2-bis(p-ethylphenyl)-
(toxicology of)

L71 ANSWER 27 OF 28 HCAPLUS COPYRIGHT 2003 ACS

AN 1958:98396 HCAPLUS

DN 52:98396

OREF 52:17371e-i,17372a-b

TI Decumbin, a new compound from a species of Penicillium

AU Singleton, V. I.; Bohonos, N.; Ullstrup, A. J.

CS Purdue Univ., Lafayette, IN

SO Nature (1958), 181, 1072-3

DT Journal

LA Unavailable

CC 11C (Biological Chemistry: Microbiology)

AB Penicillium decumbens isolated during a microbiol. study of corn spoilage in storage produced cryst. bodies which when purified and isolated resulted in colorless, odorless needles melting at 204. degree.. The compd. is considered to be a previously unreported dihydroxy, alpha.-beta.-unsatd. lactone and has been called decumbin. Production of visible crystals was best on a medium of potato-dextrose broth prep. by steaming diced, peeled potatoes in 500 ml. tap H₂O for 30 min., draining through one layer of cheese cloth, adding this to 20 g. com. glucose and dilg. to 1 l. Stationary surface culture was used since decumbin produced in submerged culture was more difficult to purify. The sterile potato-dextrose broth, in 300 ml. portions per rectangular 2 qt. milk bottle, was inoculated with a heavy spore suspension of P. decumbens.

In about 6 days at 23-26.degree., growth appeared complete with uniform grey-green sporulation and orange droplets of fluid on the surface of the mycelium. Needles of decumbin up to 1/4 in. long attached to the submerged side of the mycelium began to appear after 8 days. Cultures were usually harvested 10-12 days. One hundred bottles were strained through two layers of cheese cloth, and the mycelium extd. with 3 l-l. portions of boiling MeOH. The ext. was chilled to 10.degree., the ppt. extd. with 200-300 ml. hot MeOH. The combined exts. were chilled overnight, filtered, dild. with 100 ml. distd. H₂O and concd. in vacuo until free of MeOH. The crystals of decumbin were filtered from the aq. soln. Average yield of crude decumbin, m.p. 190.degree. was 278 mg./l. of medium in a total of 1,404 bottles processed. The crystals were purified by washing with petroleum ether (3 ml./gm.) and recrystd. from 50% (v/v) aq. MeOH (33 ml./g.) and ethyl acetate (60 ml./g.). The pure decumbin produced by several recrystns. from these two solvents was not altered by careful sublimation in vacuo or drying in vacuo over phosphoric anhyd. at 100.degree.. The solubility of decumbin at 26.degree., detd. by wt. loss from a single crystal after 19 hr. with frequent stirring, was 0.6. mg./l. of H₂O, 1.2 mg./l. of 9.5% EtOH and 24.9 g./l. of absolute EtOH. Preliminary tests showed decumbin to be **toxic to rats** and goldfish and inhibitory to wheat germination, but not an active antibiotic under **test** conditions. It gave no zones of inhibition when 1 mg./l. solns. in 40% (v/v) MeOH in H₂O were used in agar cup diffusion **tests** against *Staphylococcus aureus*, *Escherichia coli*, *Hormodendrum cladosporoides*. The acute oral L.D.50 for **rats** was of the order of 275 mg./kg. **Autopsy of rats** showed congestion of the auricles and assocd. blood vessels with dark blood and fullness of the **stomach** with undigested food. Hydrogenated decumbin was not **toxic to** two **rats** at 2,700 mg./kg. This physiol. activity or **toxicity** of decumbin in mammals, cold-blooded animals, and plants is similar to that found with other unsatd. lactones and the loss of **toxicity** upon hydrogenation is also typical of these compds.

IT Wheat
 (decumben **toxicity** to)
 IT *Penicillium decumbens*
 (dihydroxy .alpha.,.beta.-unsatd. lactone in)
 IT Lactones
 (dihydroxy, .alpha.,.beta.-unsatd., from *Penicillium decumbens*)
 IT 20350-15-6, Decumbin
 (from *Penicillium decumbens*)

L71 ANSWER 28 OF 28 HCAPLUS COPYRIGHT 2003 ACS

AN 1955:70774 HCAPLUS

DN 49:70774

OREF 49:13505b-d

TI Influence of **diet** on the acute **toxicity** of chlorpromazine in **mice**

AU Courvoisier, Simone; Cosar, Charles

SO Compt. rend. (1955), 240, 2026-7

DT Journal

LA Unavailable

CC 11H (Biological Chemistry: Pharmacology)

AB **Mice** were maintained on a balanced synthetic **diet** (I) or on an empirical **diet** (II) in order to **test** the effect of **diet** on the 50% lethal doses of chlorpromazine (2-chloro-10-(3-dimethylaminopropyl)phenothiazine-HCl) after subcutaneous administration. The L.D.50 on **diet** I for **mice** between

the ages of 90 and 100 days and with a mean wt. of 19.9 g. was 0.43 g./kg.; for mice in the same age group with a mean wt. of 24.1 g. on diet II (bread, whole milk, corn and lettuce, ad libitum) the L.D.50 was 0.175 g./kg. For mice between the ages of 53 and 55 days with a mean wt. of 16.5 g. the L.D.50 on diet I was above 0.35 g./kg.; for mice in the same age group with a mean wt. of 20.4 g. the L.D.50 was 0.16 g./kg. on diet II. For mice between the ages of 50 and 56 days with a mean wt. of 22 g., the L.D.50 on diet I was 0.4 g./kg.; for mice in the same age group with a mean wt. of 15 g. the L.D.50 was 0.2 g./kg. on diet II. For mice between the ages of 35 and 40 days with a mean wt. of 18.7 g., the L.D.50 on diet I was 0.465 g./kg.; for mice in the same age group with a mean wt. of 18.9 g. the L.D.50 was 0.18 g./kg. on diet II. These results indicate that regardless of the age or wt. of the mice the toxicity of chlorpromazine is at least twice as great on an empirical diet as on a balanced diet

IT Diet

(chlorpromazine toxicity and)

IT 50-53-3, Phenothiazine, 2-chloro-10-(3-dimethylaminopropyl)-
(therapy with)IT 50-53-3, Phenothiazine, 2-chloro-10-(3-dimethylaminopropyl)-
(toxicity of, diet and)